OBJECTIVES Elevated remnant-lipoprotein cholesterol (RLP-C) levels are associated with an increased risk of ischemic heart disease. The concurrence of RLP-C measurement by different separation methods is not well-described. This analysis assessed RLP-C by 3 commonly used measurements including immunoseparation (IM [ApoA-I and ApoB-100 monoclonal antibodies]), vertical auto profile (VAP [IDL+$VLDL_3$]) and Calculated RLP-C (Total cholesterol minus HDL-C minus LDL-C) methods using samples from a previously reported randomized, clinical trial.

METHODS This analysis assessed fasting RLP-C in hyperlipidemic patients (n=2,382) treated with ezetimibe/simvastatin (E/S)10/20 mg, E/S + niacin (N) 2g and N 2g during 24 weeks, and E/S 10/20 mg and E/S + N 2g during 64 weeks. RLP-C levels, change from baseline and % change from baseline were evaluated by the IM, VAP, and Calculated methods. The relationships and agreement among the 3 methods used in the measurement of these parameters were assessed by Pearson correlation coefficients and Bland-Altman, respectively.

RESULTS Cholesterol mass at baseline measured by the VAP and Calculated methods was ~3-4X higher than by IM; all declined with treatment by 24 weeks with little further reduction at 64 weeks (see table). RLP-C change and % reduction from baseline were larger when measured by VAP versus Calculated and IM methods. Although the 3 methods were moderately to strongly correlated (r=0.37-0.79) for RLP-C levels and changes, Bland-Altman plots showed little agreement between the methods for RLP-C levels but slightly better agreement for RLP-C changes (not shown).

CONCLUSIONS RLP-C defined by IM, VAP and Calculated methods differs in mass and response to pharmacologic intervention. Given the relationship between RLP-C and IHD risk, standardization of methods is needed for RLP-C use in risk assessment.

GW26-e2366

Changes in Carotid Plaque Lipid Content in Subjects Who Continued and Discontinued Statin Therapy

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OBJECTIVES Changes in carotid plaque lipid-rich necrotic core (LRNC) as assessed by magnetic resonance imaging (MRI) were investigated in subjects who continued and discontinued statin therapy for 2 years after a prospective study.

METHODS The Rosuvastatin Evaluation of Atherosclerotic Chinese Patients (REACH) study in 32 lipid treatment naïve subjects with LRNC showed a significant reduction in LRNC during 24 months (M) of rosuvastatin therapy. All subjects received a clinical follow-up (F/U) visit and a repeat carotid MRI scan at 48 M as planned REACH-F/U. Despite receiving a strong recommendation to continue the statin therapy at 24 M when REACH was completed, only 15 subjects continued taking statins (rosuva.=9, simva.=4 and atorva.=2) in REACH-F/U and 17 discontinued. Lipids and LRNC, both in volume (V) and % (LRNC-V/Wall V×100%), were compared between the statin-continued and -discontinued groups at 48 M.

RESULTS There were no significant differences in demographic, clinical characteristics, lipids and plaque changes during 24 M in REACH between the statin-continued and -discontinued groups in REACH-F/U. Not surprisingly, at 48 M, Total-Cholesterol (C), LDL-C and triglycerides were significantly lower in subjects who continued statin than those discontinued (163±43 vs. 207±30 mg/dl, p=0.002), (93±36 vs. 131±22 mg/dl, p=0.001) and (85±27 vs. 143±65 mg/dl, p=0.003), while HDL-C levels were similar. LRNC-V and %LRNC decreased significantly from 24 M in the statin-continued group (101±76 mm^3 at 24 M vs. 76±65 mm^3 at 48 M, p=0.001) and (17.3±11.9% at 24 M vs. 12.6±7.6% at 48 M, p=0.04). By contrast, subjects who discontinued statin showed non-statistically significant increase in LRNC-V and %LRNC (103±93 mm^3 at 24 M vs. 112±106 mm^3 at 48 M, p=0.4) and (15.4±11.3% at 24 Mvs. 16.7±11.4% at 48 M, p=0.07). Furthermore, the changes in LRNC-V and %LRNC from 24 to 48 M were significantly different between the statin-continued and -discontinued groups in REACH-F/U (-25±18 vs. 9±14 mm^3, p<0.001) and (-4.6± 8.2% vs. 1.3±2.8%, p=0.009).

CONCLUSIONS Continued statin therapy leads to continued decrease in LRNC, which indicates improved plaque stability. The REACH-F/U results provided vascular biological evidence to strongly support long-term statin therapy.

GW26-e3950

Uninterrupted Dabigatran versus Warfarin in the Treatment of Intracardiac Thrombus in Patients with non-Valvular Atrial Fibrillation

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OBJECTIVES The oral direct thrombin inhibitor dabigatran has a predictable anticoagulant effect and may be an alternative medication to warfarin for non-valvular atrial fibrillation (AF) patients with intracardiac thrombus. The objective is to compare the dabigatran, administered at a fixed dose of 150 mg twice daily (bid) with dose-adjusted warfarin (with a target international normalized ratio INR level of 2.0 to 3.0).

METHODS In the trial, 41 patients who had intracardiac thrombus detected by transesophageal echocardiography (TEE) were enrolled. Among them, 19 patients received dabigatran 150 mg bid and the remaining 22 patients received warfarin based on the patients' individual choice. Repeated TEE was performed at 3 months. The patients was assessed after 1 month and then 3 months in the clinic; meanwhile they were requested to contact the investigator immediately if symptoms developed that were suggestive of stroke, thromboembolism or major bleeding. All statistical analyses were conducted with SPSS Statistics version 17.0 software. The thrombus dissolution ability, represented by the ratio of decreased thrombus area to original area, was compared by Wilcoxon W test between the 2 groups. Difference with p value < 0.05 (2-sided) was considered statistically significant.

RESULTS Mean age of the study population was 57.7±7.4 years, with 36 (87.8%) male and 17 (41.5%) patients who had persistent AF, with no differences between the 2 groups. Thrombus area ranges from 0.1 to 4.48 cm^2 and the locations of thrombi were mainly in left atrial appendage (LAA). The thrombus area in warfarin group were larger than in dabigatran group (1.45±1.04 vs. 0.64±0.54, p < 0.05). Mean number of INR examination values obtained in the warfarin group was 10 during the therapy course. Time in therapeutic range of INR (TTR) was above 60%. Complete thrombus resolution was documented by repeated TEE in 17 patients in dabigatran group (17 of 19) and 17 patients in warfarin group (17 of 22). The ability of thrombus dissolution, represented by the ratio of decreased thrombus area to original area, was similar between the 2 groups (p > 0.05). Any bleeding, occurred in 7 patients receiving dabigatran and in 8 patients receiving warfarin. No major or fatal bleeding occurred in both two groups. One patient in the warfarin group experienced ischemic stroke. Four patients in the dabigatran group had gastrointestinal discomfort. Only one patient discontinued dabigatran for about two weeks and needed drug intervention, and after 3-month anticoagulation, a secondary TEE detected an increased thrombus.

CONCLUSIONS Dabigatran has similar effect compared with warfarin for the treatment of intracardiac thrombus in patients with non-valvular AF. Uninterrupted dabigatran is particularly essential and crucial for fibrinolysis and drug discontinuance would affect thrombus dissolution effect. It should be noticed that dabigatran lead to gastrointestinal discomfort event.

GW26-e0675

Comparison of Ticagrelor with Clopidogrel in the Treatment of Patients with Acute Coronary Syndrome in Platelet Reactivity

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OBJECTIVES To compare the inhibitory effect of ticagrelor and clopidogrel on the platelet of patients with acute coronary syndrome(ACS) after percutaneous coronary artery intervention(PCI).

METHODS 255 cases of patients with ACS admitted in our hospital from March 2014 to August 2014 were selected for this study, in which 85 cases were treated by ticagrelor and aspirin and the other 170 cases were treated by clopidogrel and aspirin respectively. All the patients were given PCI treatment, and the thrombelastography(TEG) were detected 2 days after PCI and oral administration of load dosage of antiplatelet drugs, the platelet inhibition ratio through ADP and AA pathway were observed and compared between two groups.

RESULTS Adenosine diphosphate(ADP)-induced platelet inhibition ratio in clopidogrel group was significantly lower than that of in ticagrelor group (66.60±25.57% vs 82.10±18.87%, P<0.05). Arachidonic acid(AA)-induced platelet inhibition ratio in clopidgrel was similar to that of in ticagrelor group (88.70±23.89% vs 90.32±18.09%, P>0.05). There were significant differences between clopidogrel